

Amendments to the Claims:

This listing of claims will replace all prior versions and listings of claims in the application.

Listing of Claims:

1. (currently amended) An inactive Ca^{2+} /calmodulin-dependent protein kinase II α (CaMKII α) knockin nonhuman animal, wherein a CaMKII α gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKII α is expressed, wherein lysine corresponding to Lys-42 which has at least one amino acid residue modified in the catalytic domain of mouse CaMKII α is substituted by arginine, is expressed; and thereby a protein kinase activity of CaMKII α is specifically impaired while a calmodulin binding capacity of CaMKII α and a capacity of multimerizing subunits are maintained, and wherein the inactive CaMKII α knockin nonhuman animal is produced by a gene targeting method.

2. (currently amended) The inactive CaMKII α knockin nonhuman animal of claim 1, wherein the inactive CaMKII α knockin nonhuman animal's brain nucleus accumbens has lower neuronal activity as compared to that of a wild-type, while there is no substantial difference in neuronal activities in the cerebral cortex and caudate-putamen corpus striatum as compared to those of the wild-type.

3-6. (canceled).

7. (currently amended) The inactive CaMKII α knockin nonhuman animal of claim 1 or 2, wherein the inactive CaMKII α knockin nonhuman animal is a rodent animal.

8. (previously presented) The inactive CaMKII α knockin nonhuman animal of claim 7, wherein the inactive CaMKII α knockin nonhuman animal is a mouse.

9. (currently amended) An inactive Ca^{2+} /calmodulin-dependent protein kinase II α (CaMKII α) knockin cell, wherein a CaMKII α gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKII α is expressed, wherein lysine corresponding to Lys-42 which has at least one amino acid residue modified in the catalytic domain of mouse CaMKII α is substituted by

arginine, is expressed; and thereby a protein kinase activity of CaMKII α is specifically impaired while a calmodulin-binding capacity of CaMKII α and a capacity of multimerizing subunits are maintained, and wherein the inactive CaMKII α knockin cell is produced by a gene targeting method.

10-23. (canceled)

24. (new) The inactive CaMKII α knockin cell of claim 9, wherein the cell is a rodent cell.

25. (new) The inactive CaMKII α knockin cell of claim 24, wherein the cell is a mouse cell.